RADIATION AND LIFE*

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LIFE on earth has developed with an ever-present background of radiation. It is not something new, invented by the wit of man; radiation has always been there. An issue often debated is whether life has evolved in spite of the potential deleterious effects of radiation—the winner in a constant battle—or whether the ability of radiation to cause mutations has been a vital factor in the continued upward evolution of biological species. No one is sure at present which is the case, and it is probable that the answer will never be known with any certainty.

What is new, what is man-made, is the extra radiation to which we are subjected from medical x rays in the hospital or dentist's office, from journeys in high-flying jet aircraft, from the fallout of nuclear weapon testing, from nuclear reactors built to generate electrical power, and from low level radioactive waste. There can be no denying that a man-made component of radiation is being continuously added to the background level which we receive naturally. This is a cause of great concern to the public, and must ultimately implicate the whole of society because of the critical choices and issues involved.

Many of the pollutants that we face as a by-product of this technological age are new and unique in the sense that no creature, human or otherwise, has ever had to contend with them before. For instance, many chemicals used as food additives or pesticides, much of the smoke and products of burning coal and oil, did not exist on earth in significant quantities until man made them. They are a totally new hazard faced by mankind and of his own making. No animal in its natural habitat has ever continuously inhaled smoke and the product of combustion. This is a new experience reserved for the factory workers of the 19th century and every city dweller of the twentieth.

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Radiation is not like this. It has always been present. What we are doing nowadays is adding to the existing background an extra dose of radiation from man-made devices. There is no difference in kind between natural and man-made radiations. Radiation essentially differs from other forms of pollution and this difference could turn out, in the final analysis, to be vital. Biological systems have a remarkable capacity to adapt to situations to which they are gradually exposed for a long period of time.

The initial confrontation with a new toxic agent may be devastating, but the effect is diluted as the organism adapts and evolves. Life in all forms on earth has evolved from the dawn of time against a continual background of radiation, and there is every reason to believe that living things are well able to cope, provided the levels are not too high. Repair mechanisms exist from the cell to the whole organism. This subject is discussed further by Hall.[1]

Early and Late Effects of Radiation

The immediate or acute effects of large doses of whole-body radiation have been investigated in detail in laboratory animals, and have been observed in the limited number of humans involved in nuclear accidents, most recently at Chernobyl. These effects result largely from cell killing in a critical cell population, and are only observed after doses of several Gy (several hundred rads). By contrast, late effects are due to damage to cells that survive but retain some legacy of the radiation exposure. The cell is changed in some way, and this change is passed on to the cell's progeny. If the cell concerned is a germ cell, the result may be a genetic mutation expressed in a future generation. If the cell damaged is a somatic cell, the consequence may be leukemia or cancer in the individual exposed.

Genetic effects and carcinogenesis are said to be stochastic effects. A stochastic effect is one that might arise from the injury of a few cells, or even a single cell, and therefore has no dose threshold. Any dose, however small, will carry with it some (correspondingly small) probability of producing the effect. A stochastic effect, such as cancer or a genetic mutation, is an all-or-none effect for the individual. Increasing radiation dose does not increase the severity of the effect in the individual, but does increase the frequency or incidence of the effect in a population.

Radiation Induced Cancer

Cancer induction is the most important somatic effect of low dose ionizing radiation. Information on risk estimates for leukemogenesis and carcinogen-

esis do not rely on animal data but can be based on experience in humans. There is a long history of a link between radiation exposure and an increased incidence of cancer. The human experience of carcinogenesis includes the survivors of Hiroshima and Nagasaki, patients exposed to medical irradiation, and early workers exposed occupationally. Some examples include leukemia and solid tumors among Japanese survivors, leukemia among patients irradiated for ankylosing spondylitis, thyroid cancer in children irradiated for enlarged thymus or epilated for tinea capitis, breast cancer among patients treated with x-rays for postpartum mastitis and fluoroscoped repeatedly during management of tuberculosis, lung cancer in uranium miners who breathed radon, and bone cancer among dial painters who ingested radium or patients who had injection of radium for tuberculosis or ankylosing spondylitis. Radiation carcinogenesis is a stochastic late effect. There is no threshold. It is an all-or-nothing effect; that is, the severity of the biological response is not dose related, but the probability of a response occurring is. Leukemia has the shortest latency, about five years, while solid tumors have a latency of 20 to 40 years.

Risk estimates involve an extrapolation from high doses where data are available, to low doses, which are of public health interest. Early reports of UNSCEAR and the BEIR Committee used a linear extrapolation from high to low doses. The BEIR III Committee considered a linear-quadratic model as well, which reduces risk estimates at low doses for a given observed effect at high doses.

Leukemia and bone cancer follow an absolute risk model—a discrete dose-related "crop" of radiation-induced cancer over and above the spontaneous level. It is not yet certain whether other cancers follow a relative risk model—the natural incidence increased by a constant factor. Since natural cancer incidence increases with age, this model would predict a large number of excess cancers late in life following irradiation.

There are no irradiated human population studies that follow individuals to the end of their life span, thus it is not known if absolute or relative risk models apply (except for leukemia and bone cancer, which appeared in excess for a number of years after irradiation and then returned to spontaneous levels, i.e., follow an absolute risk model). The best risk estimates are for leukemia, thyroid cancer, and breast cancer. Others (e.g., lung and bone) are subject to greater uncertainty.

Some representative values are listed in Table I. The total cancer risk for total-body irradiation is about one death per 10,000 individuals exposed to 1 rem (0.01 Sv). For every leukemia, three to four solid tumors are induced in

TABLE 1. REPRESENTATIVE RISK ESTIMATES FOR CANCER*

Type or site of cancer	*Risk/10^6/rem, or risk/10^4/Sv*
*Fatal**	
Leukemia	20
Thyroid	5
Breast	25
Lung	20
Bone	5
Liver	10
Lower large intestine	10
Skin	1
Others	30
All cancers	about 100
Nonfatal	
Thyroid	100
Breast	25
Skin	100

*There are wide variations in risk estimates published by the BEIR III Committee or UNSCEAR, depending on the method of extrapolation used and the model for carcinogenesis assumed. None of these estimates must be taken too seriously, since the data upon which they are based are so poor and the uncertainties so large. They are no more than ballpark figures. The values in their table are largely those suggested by Sir Edward Pochin in the Sizewell B Inquiry.

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an irradiated population. Radiation induced carcinogenesis is discussed in more detail by Hall 1988[2] and by the BEIR III and UNSCEAR reports.[3,4]

GENETIC EFFECTS OF RADIATION

The fact that mutants produced by man-made radiations cannot be recognized or identified as different from natural spontaneous types makes their study particularly difficult. Sample sizes must be large to detect a small increase caused by radiation.

Few human data are available on the genetic effects of radiation, except the limited observations of genetic consequences in the children of Japanese survivors of Hiroshima and Nagasaki. An important quote from the BEIR III Report is: "The estimation of genetic risks in the human must be based almost entirely on animal data." Radiation-induced genetic changes, like mutations from any other agent, may be a consequence of gene mutation or

chromosomal changes. Radiation does not produce new, unique mutations but increases the incidence of the same mutations that occur spontaneously. First generation mutations in mice have been measured by observing skeletal anomalies in the offspring of irradiated mice. Relative mutation rates have been measured in the megamouse project by observing specific locus mutations.

The doubling dose is the dose required to double the spontaneous mutation incidence; put another way, it is the dose required to produce an incidence of mutations equal to twice the spontaneous rate. Based on the mouse data, the doubling dose for low dose-rate exposure in humans was estimated by the BEIR III Committee to be in the range of 50 to 250 rems (0.5 to 2.5 Sv). The corresponding estimate of the 1986 UNSCEAR report was 100 rads (1 Gy).

Parental irradiation at 1 rem (10 mSv) per 30-year generation results in 5 to 65 additional genetic disorders per million live-born offsprint (compared with 10,000 spontaneous) and about 50 to 1,100 per million in equilibrium if the irradiation is continued for several generations (compared with 107,000 spontaneous); that is, 1 rem (10 mSv) per generation parental exposure increases the spontaneous mutation incidence by about 1%. These data from the BEIR III Committee are summarized in Table II. Not more than 1% to 6% of spontaneous mutations in humans may be ascribed to background radiation.

TABLE II. GENETIC EFFECTS OF AN AVERAGE POPULATION EXPOSURE OF 1 REM (10 mSv) PER 30-YEAR GENERATION COMPARED WITH THE SPONTANEOUS LEVELS

Type of genetic disorder	*Spontaneous incidences per million live-born offspring*	*Effect of 1 rem (10 mSv) per generation per million live offspring*	
		First generation	*Equillibrium*
Autosomal dominant and X-linked	10,000	5-65	40-200
Irregularly inherited	90,000	5-65	20-900
Recessive	1,100	Very few; effects in heterozygotes accounted for in top row	Very slow increase
Chromosomal	6,000	Fewer than 10	Increases only slightly

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Children of the survivors of Hiroshima or Nagasaki have been studied for untoward pregnancy outcomes, death of live-born children, sex chromosome abnormalities, and electrophoretic variants of blood proteins. Though no genetic indicator is statistically significant, the average doubling dose is 158 rems (1.58 Sv). These data suggest that humans are not more sensitive and are probably less sensitive than mice to radiation-induced genetic effects. Based on the limited human data, it is concluded that the mouse data for radiation-induced genetic effects can be applied to humans with some measure of confidence. The genetic effects of radiation are discussed in more detail by Hall[2] and by the BEIR III, and UNSCEAR committees reports.[3,4]

Low Level Radioactive Waste

The term "low-level waste" serves for a very wide range of radioactive waste. Any industry, hospital, medical, educational or research institute, private or government laboratory, and other activity that utilizes radioactive material produces a low-level radioactive waste. More than 20,000 companies, institutions, laboratories, and government facilities are licensed by the Nuclear Regulatory Commission or Agreement States to use radioactive materials as a normal part of their activities. Most of these users have some form of low-level radioactive waste for disposal. The Nuclear Regulatory Commission predicted that during the period 1980-2000 about 65% of this waste will be from fuel cycle sources, and 19% of the nonfuel-cycle sources will be from medical and educational institutions.[5] More recent data based on the quantity of waste received at the commercial burial sites indicate this volume of activity to be 2.9% and 0.06%, respectively.[6]

Radiation Doses to the Public

Estimated radiation exposures to individuals living near low level radioactive waste disposal sites have been a small fraction of the annual limits of 0.25 mSv (25 mrem) to the whole body and individual organs. The principal pathway for radiation exposure to the public was inhalation of tritium from the effluent of the evaporator used to treat leachate pumped from the burial trenches.[7] For example, the collective dose equivalent commitment to the population in the vicinity of the Maxey Flats site from one year of operation of the facility was estimated to be 0.042 person-Sv (4.2 person-rem).[8]

This must, of course, be multiplied by the number of burial sites, but at the present time there are only three and will never likely exceed 10. These doses are tiny and should be viewed against the total radiation exposure of the American population.

IONIZING RADIATION EXPOSURE OF THE AMERICAN POPULATION

Natural sources make the greatest contribution to the average annual effective dose equivalent for members of the American population. Among these natural sources, radon and radon decay products indoors are the largest contributors to the average annual effective dose equivalent, and they make a small contribution to the annual genetically significant dose. Among man-made or enhanced sources, medical exposures contribute the largest exposure. These exposures differ in character however, from inadvertent exposures, in that they contribute to the benefit of the specific individual receiving them. Other people are affected only through the genetically significant dose to the population. The contribution to the population dose from most of the other sources, including nuclear power and consumer products (with the possible exception of tobacco), are minor.[9] They are summarized in Table III and Figure 1.

CONCLUSION

By any reasonable assessment, the radiation hazards to human health posed by low level radioactive waste are trivial compared with medical radiation, which in turn is dwarfed by the potential deleterious effects of radon. People who live close to a nuclear power plant, or to a burial site for radioactive waste, get far more radiation from radon exposure in their homes in one day than they get in a year from the power-plant or burial site neighbor! For the population as a whole, the annual collective effective dose equivalent for radon is at least ten million times larger than for low level radioactive waste. If we want to address a major problem in our society, the solution to which may save thousands of lives, radon would appear to be a good candidate. A number of estimates have been made of the possible number of lung cancer deaths per year in the United States attributable to radon. BEIR IV estimated 13,000,[10] while the EPA suggested a wide range of 5,000 to 20,000.[11] By no stretch of the imagination can even one cancer death per year be attributed to low-level radioactive waste.

The cost of saving a life by random remedial action is far less than by improving low level waste facilities. It has been estimated that our government is spending about $200 million per eventual life saved to protect future Americans against radiation from nuclear waste, and has required utilities to spend $2 billion per life saved to protect us from the radiation from reactor accidents. By contrast, the estimate is $10,000 to save a life by reducing radon levels.[12]

TABLE III. ANNUAL EFFECTIVE DOSE EQUIVALENT IN THE U.S. POPULATION CIRCA 1980-82

Source	*Number of people exposed (thousands)*	*Average annual Hg* in the exposed population (mSv)***	*Annual collective effective dose equivalent (person-Sv)†*	*Average annual Hg* in the U.S. population (mSv)‡*
Natural sources				
Radon	230,000	2.0	460,000	2.0
Others	230,000	1.0	230,000	1.0
Occupational	930‖	2.3	2,000	0.009
Nuclear fuel cycle	-§	-	136	0.0005
Consumer products				
Tobacco	50,000	-	-	-
Other	120,000	0.05-0.13	12,000-29,000	0.05-0.13
Miscellaneous environmental sources	~25,000	0.006	160	0.0006
Medical				
Diagnostic x rays	-	-	91,000	0.39
Nuclear medicine	-	-	32,000	0.14
Rounded total	230,000	-	835,000	3.6

*Hg is the effective dose equivalent

**1 mSv = 100 mrem.

†1 person-Sv = 100 person-rem.

‡Those nominally exposed total 1.68×10^4.

‖Collective doses were calculated to the regional population within 80 km (50 miles) of each facility.

§Effective dose equivalent to determine; dose to a segment of bronchial epithelium estimated to be 0.16 Sv/y (16 rem/y), see Section 5.3.

From NCRP 93 (reference 9)

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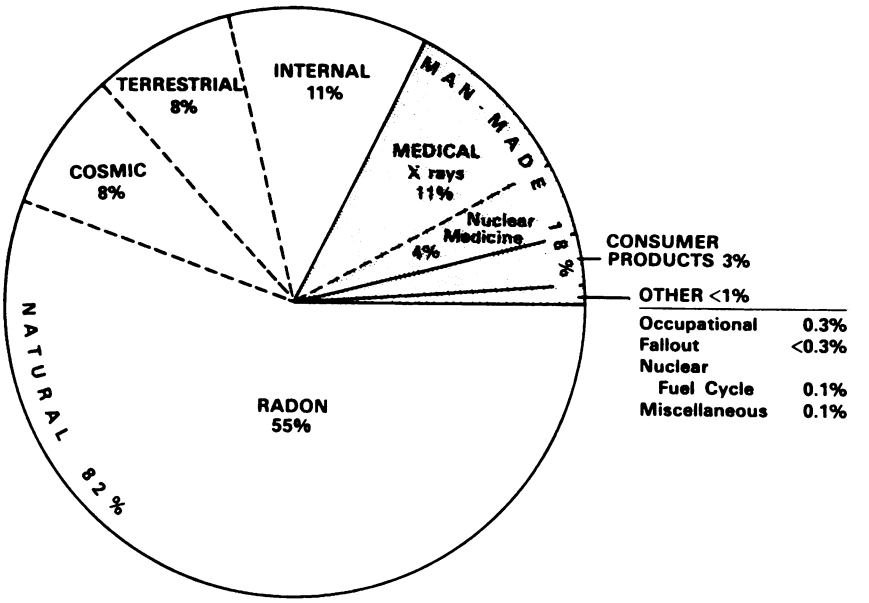

The percentage contributions of various radiation sources to the total average effective dose equivalent in the U.S. population Reproduced from reference 3. Reproduced by permission from NCRP93: *Ionization Radiation Exposure of the Population of the United States*. Bethesda, MD, National Council on Radiation Protection and Measurements, 1987.

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